

## **5.16 PUBLIC HEALTH**

This section presents the methodology and results of a human health risk assessment performed to assess potential public health impacts associated with airborne emissions from the construction and routine operation of the MPP. The analysis evaluated potential emissions of “air toxic” compounds from the turbine stack, auxiliary boiler, and cooling tower. Air toxics are compounds for which ambient air quality standards have not been established, but are known or suspected to cause short-term (acute) and/or long-term (chronic or carcinogenic) adverse health effects. “Criteria Pollutants” (compounds with ambient air quality standards) are addressed in Section 5.2 and summarized in Section 5.16.2.5. Potential exposures due to upset conditions are addressed in Section 5.15. Also of concern with respect to public health are potential exposures to electric and magnetic fields (EMF). Potential public health impacts from electromagnetic exposure are discussed in Section 5.16.3.

Air is the dominant pathway for public exposure to chemical substances that will be released by the project. Emissions to the air will consist of combustion by-products produced in the gas turbine and boiler, as well as emissions from the cooling water. Potential health risks from multiple exposure pathways, including inhalation, were addressed for identified sensitive receptors and at the points of maximum predicted inhalation exposure. The air pathway and multipathway portions of the risk assessment were conducted in accordance with guidance established by the California Air Pollution Control Officers Association (CAPCOA).

### **5.16.1 Affected Environment**

For purposes of the air quality and public health exposure assessments, it was assumed that the turbine stack will exhaust combustion gases at approximately 150 feet (45.72 meters) above grade elevation (560 feet or 171 meters). Topographical features within a ten-mile radius that are of equal or greater elevation than the assumed stack exhaust exit point (stack height plus grade elevation; 710 feet or 216 meters) are shown on Figure 5.16-1.

Sensitive receptors are defined as individuals that may be more susceptible to health risks due to chemical exposure. Schools (public and private), day care facilities, convalescent homes, and hospitals are of particular concern, since they are likely to have a concentration of sensitive receptors. The nearest sensitive receptor within a one-mile radius of the power plant site is an elementary school located approximately 0.68 kilometers (0.42 miles) southwest of the site. The nearest residence is approximately 600 feet northwest of the site on Moss Street. Potential sensitive receptors located within 5 miles of the site are shown on Figure 5.16-2.

## **5.16.2 Environmental Consequences**

### **5.16.2.1 Public Health Risks - Construction Phase**

The demolition of the remaining components associated with Magnolia Units 1 and 2 is expected to take approximately four to six months and will be completed prior to expansion construction. The construction phase of the MPP is expected to take approximately 23 months. No significant public health effects are expected during the construction phase. Strict construction practices that incorporate safety and compliance with all applicable LORS will be followed (see Section 7.0). Further, mitigation measures to reduce construction impacts will be implemented as described in Section 5.2.6.

Temporary emissions from demolition and construction-related activities are discussed in Section 5.2. Ambient air modeling for PM<sub>10</sub>, CO, and NO<sub>x</sub> was performed as described in Section 5.2.4.1 and Appendix H-2. Construction-related emissions are temporary and localized, resulting in no long-term impacts to the public. All predicted maximum concentrations occurred at locations along the immediate property boundary.

Small quantities of hazardous waste may be generated during the construction phase. Hazardous waste management plans will be in place so that the potential for public exposure will be minimal. Refer to Section 5.14 (Waste Management) for more information.

### **5.16.2.2 Public Health Risks - Operational Impacts**

The methods used to assess potential human health risks from routine operations are consistent with those presented in the document prepared by the CAPCOA, *Air Toxics "Hot Spots" Program: Revised 1992 Risk Assessment Guidelines* (CAPCOA, 1993), and SCAQMD *Guidance; Risk Assessment Procedures for Rules 1401 and 212* (SCAQMD, 2000). These guidelines were developed for the adoption of Rule 1401 (New Source Review) and Rule 212 (Standards for Approving Permits). The document provides assistance for evaluating Rule 1401 compliance. The CAPCOA guidelines provide risk assessment procedures for use in the preparation of the health risk assessments required under the Air Toxics "Hot Spots" Information and Assessment Act of 1987, AB 2588 (Health and Safety Code Section 44360 et seq.). The "Hot Spots" law established a statewide program for the inventory of air toxics emissions from individual facilities, as well as requirements for risk assessment and public notification of potential health risks.

The health risk assessment was conducted in three basic steps. First, a hazard identification was performed to determine pollutants of concern associated with facility operations. Second, an exposure assessment was performed that included toxic air contaminant emission

calculations and the simulation of pollutant transport using atmospheric dispersion modeling and multi-environmental pathway exposure and analysis. Third, a risk characterization was performed analyzing potential health risks from these calculated exposures, which included identifying the location of maximum cancer and non-cancer health risks. The multipathway analysis included the inhalation pathway, dermal (skin) absorption, ingestion of soil with deposited pollutants, plant pathway, and exposure to pollutants potentially in mother's milk. Consideration of these pathways are consistent with risk screening procedures contained in the CAPCOA Guidelines (CAPCOA, 1993) and the SCAQMD guidance (SCAQMD, 2000a; 2000b; and 2001).

**5.16.2.2.1 Hazard Identification.** The hazard identification involved an evaluation of turbine operations of the turbine, auxiliary boiler and cooling tower to determine if there are particular substances that will be used or that may be generated, which may cause negative health effects if released to the air. The chemicals evaluated in this analysis were identified from the CAPCOA guidelines (CAPCOA, 1993), the California Office of Environmental Health Hazard Assessment (OEHHA) *Technical Support Document for Describing Available Cancer Potency Factors* (Cal-EPA 1999a), OEHHA's *The Determination of Acute Reference Exposure Levels for Airborne Toxicants for Airborne Toxicants* (Cal-EPA, 1999b), and *The Determination of Chronic Reference Exposure Levels* (Cal-EPA, 2000a; 2000b; and 2001).

Table 5.16-1 presents a list of substances that may be emitted from the turbines (excluding pollutants with established ambient air quality standards, which are addressed in Section 5.2) along with their toxic effects and toxicological endpoints.

Section 5.15, Hazardous Material Handling, provides more detailed information on chemicals stored and used on site and the potential impacts associated with their use and storage. A discussion of the consequences of a potential accidental release of hazardous materials is also included in Section 5.15.

#### **5.16.2.2.2 Exposure Assessment Methods.**

##### **Significance Criteria.**

**Cancer Risk.** Cancer risk is the probability or chance of contracting cancer over a human life span (assumed to be 70 years). Carcinogens are assumed not to have a threshold below which there would be no human health impact. In other words, any exposure to a carcinogen is assumed to have some probability of causing cancer; the lower the exposure, the lower the cancer risk (i.e., a linear, no-threshold model). Under various state and local regulations, an

**TABLE 5.16-1**

**TOXIC AIR CONTAMINANTS POTENTIALLY EMITTED FROM THE EMISSION SOURCES**

<b>Pollutant</b>	<b>Turbine and Boiler</b>	<b>Cooling Tower</b>	<b>Carcinogen</b>	<b>Chronic Non-Carcinogen</b>	<b>Acute Non-Carcinogen</b>	<b>Toxicological Endpoint (Chronic Toxicity)</b>
Acetaldehyde	X		X	X		Respiratory system
Acrolein	X			X	X	Respiratory system
Ammonia	X	X		X	X	Respiratory, skin irritation or other effects
Arsenic		X	X	X	X	Development; cardio vascular system; nervous system
Benzene	X		X	X	X	Immune system, developmental nervous system
Bis (2-ethylhexyl)-phthalate		X	X	X		Alimentary system
Cadmium		X	X	X		Kidney; respiratory system
Chloroform		X	X	X	X	Alimentary system; kidney; development
Chromium		X	X	X		Respiratory (hexavalent form only; also applies to carcinogenesis)
Copper		X		X	X	Respiratory system
1,4-Dichlorobenzene		X	X	X		Nervous system; respiratory system; alimentary system; kidney
Ethylbenzene	X	X		X		Development; alimentary system; kidney, endocrine system
Formaldehyde	X		X	X	X	Respiratory system; eyes
Hexane	X			X		Nervous system
Lead		X	X			Cardiovascular system; nervous system, immune system, kidney; reproductive system
Manganese		X		X		Nervous system
Mercury		X		X	X	Nervous system
Methylene chloride		X	X	X	X	Cardiovascular system; nervous system
Methyl-t butyl ether (MTBE)		X		X		Kidney; eyes; alimentary

**TABLE 5.16-1**

**(CONTINUED)**

<b>Pollutant</b>	<b>Turbine and Boiler</b>	<b>Cooling Tower</b>	<b>Carcinogen</b>	<b>Chronic Non-Carcinogen</b>	<b>Acute Non-Carcinogen</b>	<b>Toxicological Endpoint (Chronic Toxicity)</b>
Nickel		X	X	X	X	Respiratory system; immune system
Naphthalene	X			X		Respiratory system
PAHs	X	X	X			No listed non-carcinogenic effects (Human carcinogen)
PCBs		X	X	X		Immune system; alimentary system; reproductive system
Phenol		X		X	X	Alimentary system; cardiovascular system; kidney; nervous system
Propylene	X			X		Respiratory system
Selenium		X		X		Respiratory system
Tetrachloroethylene		X	X	X	X	Alimentary system; kidney
Toluene	X	X		X		Central or peripheral nervous system, respiratory system, and reproductive system including teratogenic and developmental effects
Xylene	X			X	X	Nervous system and respiratory system
Zinc		X		X		Respiratory system; cardiovascular system

incremental cancer risk of 10-in-one-million as the result of a project is considered to be a significant impact on public health. For example, the 10-in-one-million risk level is used by the Air Toxics “Hot Spots” (AB 2588) program and California’s Proposition 65, as the public notification level for air toxic emissions from existing sources. The SCAQMD allows for an incremental risk of 10-in-one-million in permitting new sources provided toxics best available control technology (T-BACT) is employed, which for combustion sources is generally considered to be the firing of natural gas. For assessing the significance of potential risks from the MPP emissions, a significant impact criteria for lifetime incremental cancer risk of 10-in-a-million is appropriate. The CEC generally does not consider potential mitigation measures if calculated maximum cancer risks are less than one-in-one-million.

The lifetime risk of cancer from all causes combined is about 400,000 in a million (or about 40 %) in the United States today (National Cancer Institute [NCI], 2000). Environmental and occupational exposures are generally thought to be responsible for a small portion of this background risk. However, environmental and occupational carcinogens are a principal focus of regulatory policy because they are often involuntary, and in principle can be reduced by regulatory initiatives. The project’s maximum incremental risk will not appreciably change the lifetime risk at receptors in the area, as discussed in Section 5.16.2.3.

**Non-Cancer Risk.** Non-cancer health effects can be either chronic or acute. In determining potential non-cancer health risks (chronic and acute) from air toxics, it is assumed that there is a dose of the chemical of concern below which there would be no impact on human health. In other words, there is a threshold below which no effects occur. The air concentration corresponding to this dose is called the reference exposure level (REL), and for the non-inhalation environmental pathways, the threshold dose is typically expressed in terms of the reference dose (RfD), which is an allowable daily dose per body weight (mg/kg-day). Non-cancer health risk is measured in terms of a hazard quotient, which is the calculated exposure of each contaminant divided by its REL. Hazard quotients for those pollutants that affect the same target organ are typically summed, and the resulting totals expressed as hazard indices for each organ system. A hazard index of less than 1.0 is considered to be an insignificant health risk. The acute RELs used in the hazard index calculations were those published by OEHHHA in March 1999 (Cal EPA, 1999b). The chronic RELs used were those updated by OEHHHA in February 2000, April 2000, and January 2001 (Cal-EPA, 2000a; 2000b; and 2001). Any chronic REL not updated by OEHHHA was obtained from the CAPCOA Guidelines (CAPCOA, 1993).

Chronic toxicity is defined as adverse health effects from prolonged chemical exposure, and is caused by chemicals accumulating in the body. Since chemical accumulation to toxic levels typically occurs slowly, symptoms of chronic effects usually do not appear until long after

exposure commences. The lowest no-effect chronic exposure level for a non-carcinogenic air toxic is the chronic REL or RfD. Below these thresholds, the body is capable of eliminating or detoxifying the chemical rapidly enough to prevent its accumulation. The chronic hazard index was calculated using the hazard quotients calculated with annual concentrations.

Acute toxicity is defined as adverse health effects caused by a brief chemical exposure of no more than 24 hours. For most chemicals, the air concentration required to produce acute effects is higher than levels required to produce chronic effects because the duration of exposure is shorter. Acute toxicity is predominantly manifested in the upper respiratory system at threshold exposures. One-hour average concentrations are divided by acute RELs to obtain a hazard index for health effects caused by relatively high, short-term exposure to air toxics.

**Air Toxic Emissions.** The potential emissions of air toxic compounds from the turbine and auxiliary boiler were assessed using air toxic emission factors for combustion sources obtained from the SCAQMD. These emission factors were developed for AB 2588 Toxic “Hot Spots” source test data by the SCAQMD.

Consistent with modeling performed for criteria pollutants (Section 5.2), annual emissions were calculated assuming 1,000 hours per year of operations with duct firing and assuming no duct firing for the remainder of the year (assuming 8,760 hours per year at 100 percent load). This was used for the annual average emissions estimates for the calculation of carcinogenic and chronic non-cancer health effects. For acute non-cancer health impacts, maximum hourly emissions were used assuming operation of the turbine at 100 percent load during duct firing at 95° F. Emission rates are summarized in Table 5.16-2 and 5.16-3. Ammonia slip emission rates are included in these emission estimates based on an allowable ammonia slip level of 5 parts per million per dry volume (ppmdv) in the turbine stack gas, corrected to a 15 percent oxygen level. These ammonia emission calculations are outlined in Appendix H-13.

Emissions of trace elements from cooling tower drift were estimated based on measured water quality data for the make-up water to be used in the cooling tower. These data were combined with the expected operational data for the cooling towers (e.g., drift rate, water circulating rate, and cycles of concentration) to estimate emissions of toxic compounds. Engineering judgement was applied to assume that any chlorides present in the make-up water will remain in solution as salts and not emitted in hazardous form. Further, the conservative decision was made to assume that all chromium present would be hexavalent chromium. Emissions of toxics from the cooling towers are presented in Table 5.16-4.

**TABLE 5.16-2**

**EMISSION RATES FOR COMBUSTION TOXIC AIR CONTAMINANTS**

<b>Pollutant</b>	<b><u>Emission Factors (lb/MM ft<sup>3</sup>)</u></b>		<b><u>Emission (tons/yr)</u></b>		<b><u>Emission (lbs/hr)</u></b>	
	<b>Turbine<sup>2</sup></b>	<b>Boiler<sup>3</sup></b>	<b>Turbine<sup>2</sup></b>	<b>Boiler<sup>3</sup></b>	<b>Turbine<sup>2</sup></b>	<b>Boiler<sup>3</sup></b>
Acetaldehyde	0.037	0.0043	2.851E-01	1.141E-04	6.894E-02	2.605E-05
Acrolein	0.009	0.0027	6.935E-02	7.163E-05	1.677E-02	1.635E-05
Ammonia <sup>1</sup>	--	--	5.247E+01	--	1.293E+01	--
Benzene	0.0113	0.008	8.707E-02	2.122E-04	2.105E-02	4.846E-05
Ehtylbenzene	0.0132	0.0095	1.017E-01	2.520E-04	2.459E-02	5.754E-05
Formaldehyde	0.094	0.017	7.243E-01	4.510E-04	1.751E-01	1.030E-04
Hexane	1.75	0.0063	1.348E+01	1.671E-04	3.260E+00	3.816E-05
Naphthalene	0.0008	0.0003	6.164E-03	7.959E-06	1.491E-03	1.817E-06
PAHs	0.001	0.0004	1.541E-03	2.653E-06	3.726E-04	6.057E-07
Propylene	1.0522	0.731	8.108E+00	1.939E-02	1.960E+00	4.428E-03
Toluene	0.0726	0.0366	5.594E-01	9.710E-04	1.353E-01	2.217E-04
Xylenes	0.0298	0.0272	2.296E-01	7.216E-04	5.552E-02	1.648E-04

<sup>1</sup> For annual, assume no hours of steam injection because ammonia slip is lower for steam injection.

For hourly, assume 41° F (100% load) case since ammonia slip is higher at this temperature.

<sup>2</sup> Emission factors from SCAQMD Web Site (<http://aqmd.gov/permit/comb.html>)

Internal combustion - Turbine.

<sup>3</sup> Emission factors from SCAQMD Web Site (<http://aqmd.gov/permit/comb.html>)

External combustion < 10MM Btu/hr.



**TABLE 5.16-3**

**TURBINE TOXIC AIR CONTAMINANT EMISSIONS USED IN  
ATMOSPHERIC DISPERSION MODELING**

<b>Pollutant</b>	<b><u>Annual Emission (g/s)</u></b>		<b><u>Hourly Emission (g/s)<sup>1</sup></u></b>	
	<b>Turbine</b>	<b>Boiler</b>	<b>Turbine</b>	<b>Boiler</b>
Acetaldehyde	8.20E-03	3.28E-06	8.69E-03	3.28E-06
Acrolein	1.99E-03	2.06E-06	2.11E-03	2.06E-06
Ammonia	1.51E+00	----	1.63E+00	----
Benzene	2.50E-03	6.11E-06	2.65E-03	6.11E-06
Ehtylbenzene	2.93E-03	7.25E-06	3.10E-03	7.25E-06
Formaldehyde	2.08E-02	1.30E-05	2.21E-02	1.30E-05
Hexane	3.88E-01	4.81E-06	4.11E-01	4.81E-06
Naphalene	1.77E-04	2.29E-07	1.88E-04	2.29E-07
PAHs	4.43E-05	7.63E-08	4.70E-05	7.63E-08
Propylene	2.33E-01	5.58E-04	2.47E-01	5.58E-04
Toluene	1.61E-02	2.79E-05	1.70E-02	2.79E-05
Xylenes	6.61E-03	2.08E-05	7.00E-03	2.08E-05

Emission calculations based on the Westinghouse 501F

<sup>1</sup> Because the turbine and boiler will not operate simultaneously and because turbine erosions are much higher, the boiler was not included in the acute modeling analysis.

**TABLE 5.16-4****COOLING TOWER TOXIC AIR CONTAMINANT EMISSIONS**

<b>Pollutant</b>	<b>(g/s)</b>	<b>lb/hr</b>	<b>TPY</b>
Ammonia	5.32E-03	0.0422	0.185
Arsenic	5.91E-07	0.0000047	0.000021
Bis(2-ethylhexyl)-phthalate	1.68E-05	0.00013	0.00059
Cadmium	1.97E-06	0.000016	0.000068
Chloroform	1.32E-06	0.000010	0.000046
Chromium	1.97E-06	0.000016	0.000068
Copper	2.28E-06	0.000018	0.000079
1,4-dichlorobenzene	5.91E-07	0.0000047	0.000021
Ethylbenzene	9.85E-08	0.00000078	0.00013
Lead	9.85E-06	0.000078	0.00034
Manganese	5.32E-06	0.000042	0.00018
Mercury	3.94E-08	0.00000031	0.0000014
Methylene chloride	5.91E-07	0.0000047	0.000021
MTBE	2.96E-07	0.0000023	0.000010
Nickel	1.97E-06	0.000016	0.000068
PAH	7.88E-07	0.0000063	0.000027
PCB	3.94E-08	0.00000031	0.0000014
Phenol	5.91E-06	0.000047	0.00021
Selenium	3.94E-07	0.0000031	0.000014
Tetrachloroethylene	9.85E-08	0.00000078	0.0000034
Toluene	9.85E-08	0.00000078	0.0000034
Zinc	2.57E-05	0.00020	0.00089

**Dispersion Modeling Methodology.** Atmospheric dispersion modeling was performed to estimate offsite, ground-level concentrations of toxic air contaminants that may be emitted due to operation of the turbine, auxiliary boiler and cooling tower. Modeling methodologies follow those discussed for the refined modeling analysis in Section 5.2. The USEPA-approved ISCST3 model was used to estimate these ground-level concentrations in all terrain settings based on one year (1981) of hourly meteorological data collected in Burbank by the SCAQMD. Upper air data used for daily mixing heights were also supplied by the SCAQMD and calculated from Ontario upper air data.

To identify the points of maximum impact, a multi-scale grid of receptors was used in the ISCST3 modeling. Near the MPP site, receptors were placed along the property boundary at approximately 25-meter increments. Additional receptors were placed in 50-meter increments to a distance of 0.5 kilometers, at 100-meter increments to a distance of 1 kilometer, and at 250-meter increments to a distance of 10 kilometers. Sensitive receptors were included to a distance of 5 miles. A list of sensitive receptors can be found in Appendix P (EDR, 2001).

The ISCST3 modeling results were then incorporated in the health risk analysis in the AB 2588 model. AB 2588 uses an ISCST3 binary output in conjunction with source emission rates and toxicity factors, to calculate human health effects. For cancer risk, estimated ground-level concentrations of each substance (in micrograms per cubic meter [ $\mu\text{g}/\text{m}^3$ ]) were multiplied by its cancer “unit risk factor”, which is the estimated cancer risk for a continuous exposure to  $1 \mu\text{g}/\text{m}^3$  over a specified averaging time, usually assumed as 70 years in a lifetime cancer risk estimate. The cancer unit risk factors were obtained from the updated OEHHA *Technical Support Document for Describing Available Cancer Potency Factors* (Cal-EPA, 1999a). Table 5.16-5 summarizes cancer unit risk factors used in the health risk assessment modeling.

For chronic non-cancer health effects, calculated annual exposures were divided by pollutant-specific chronic RELs published by OEHHA (Cal-EPA 2000a; 2000b; and 2001) and CAPCOA (1993), and summed by the AB 2588 model per affected target organ, to calculate a chronic hazard index. For acute non-cancer health effects, calculated maximum hourly exposures were divided by pollutant-specific acute RELs published by OEHHA (Cal-EPA, 1999b), and summed by the AB 2588 model per affected target organ, to calculate an acute hazard index. Table 5.16-5 summarizes chronic and acute non-cancer RELs used in the health risk assessment modeling.

Electronic input and output files for the ISCST3 dispersion modeling and AB 2588 health risk runs will be submitted on CD-ROM to the CEC and the SCAQMD under separate cover.

**TABLE 5.16-5**  
**TOXICOLOGICAL FACTORS USED IN THE**  
**HEALTH RISK ASSESSMENT MODELING**

<b>Chemical</b>	<b>Unit Risk Factor</b>	<b>Acute Reference Exposure Level</b>	<b>Chronic Reference Exposure Level</b>
Acetaldehyde	2.70E-06	----	9.00E+00
Acrolein	----	1.90E-01	6.00E-02
Ammonia	----	3.20E+03	2.00E+02
Arsenic	3.30E-03	1.90E-01	3.00E-02
Benzene	2.90E-05	1.30E+03	6.00E+01
Bis (2-ethylehexyl-phthalate)	2.40E-06	----	7.00E+01
Cadmium	4.20E-03	----	2.00E+02
Chloroform	5.30E-06	1.50E+02	3.00E+02
Chromium (hex.)	1.50E-01	----	2.00E-01
Copper	----	1.00E+02	2.40E+00
Dichlorobenzene-P	1.10E-05	----	8.00E+02
Ethyl Benzene	----	----	2.00E+03
Formaldehyde	6.00E-06	9.40E+01	3.00E+00
Hexane	----	----	7.00E+03
Lead	1.20E-05	----	1.50E+00
Manganese	----	----	2.00E-01
Mercury	----	1.80E+00	9.00E-02
Methylene Chloride	1.00E-06	1.40E+04	4.00E+02
MethylTerButylEther	2.60E-07	----	8.00E+03
Naphthalene	----	----	9.00E+00
Nickel	2.60E-04	6.00E+00	5.00E-02
PAHs	1.10E-03	----	----
Phenol	----	5.80E+03	2.00E+02
Polychlor. Biphenyls	2.00E-05	----	1.20E+00
Propylene	----	----	3.00E+03
Selenium	----	----	5.00E-01
Tetrachloroethylene	5.90E-06	2.00E+04	3.50E+01
Toluene	----	3.70E+04	3.00E+02
Xylene	----	2.20E+04	7.00E+02
Zinc	----	----	3.50E+01

**5.16.2.2.3 Risk Characterization.** Carcinogenic risks and potential chronic and acute non-cancer health effects were assessed using the dispersion modeling described above and numerical values of toxicity recommended in the OEHHA technical support document on cancer potency factors (CalEPA, 1999a), the OEHHA update on chronic and acute RELs (CalEPA, 1999b; 2000a; 2000b; and 2001) and the CAPCOA Guidelines (1993). The environmental pathways analyzed included inhalation, dermal absorption (skin), soil ingestion, plant exposure, and exposure through mother's milk. The inhalation, dermal absorption, soil ingestion, and mother's milk pathways are recommended in the CAPCOA guidelines (1993) for a screening-level health risk assessment.

The chief exposure assumption is one of continuous exposure (at maximum emission rates) over a 70-year period at each identified receptor location. When combined with EPA-approved dispersion modeling methodologies, the use of OEHHA cancer potency factors and OEHHA and CAPCOA RELs/RfDs, provides an upper bound estimate of the true risks. That is, the actual risks are not expected to be any higher than the predicted risks and are likely substantially lower. A discussion of uncertainty factors is presented in Section 5.16.2.4.

### 5.16.2.3 Study Results

**5.16.2.3.1 Estimated Cancer Risks.** Table 5.16-6 presents the estimated lifetime cancer risk at the maximum impact point attributable to all carcinogenic contaminants from routine operations. The maximum incremental lifetime cancer risk was calculated to be approximately 0.37 in-one-million at a location approximately 1.8 kilometers north-west of the proposed project. This calculated cancer risk is below the significance criterion of 10-in-one-million. An excess cancer burden was not calculated because the maximum cancer risk is below one-in-one million, in accordance with SCAQMD health risk assessment procedures. The highest cancer risk at a sensitive receptor is 0.25 in-one-million.

**TABLE 5.16-6**

#### **HEALTH RISK ASSESSMENT RESULTS**

Maximum Cancer Risk <sup>1</sup>	0.37 in-one-million
Maximum Chronic Hazard Index <sup>1</sup>	0.023
Maximum Acute Hazard Index	0.082

<sup>1</sup> Average value at maximum impact location calculated over one year (1981) of meteorological conditions.

**5.16.2.3.2 Estimated Non-Cancer Health Effects.** Table 5.16-6 shows that the calculated chronic non-cancer hazard index at the maximum impact location attributable to the turbine emissions was calculated as 0.023 for the maximally impacted target organ system. For assessing chronic non-cancer health effects, calculated exposures were based on annual-average dispersion modeling results. Table 5.16-6 also shows a calculated acute hazard index of 0.082 at the maximum impact location. Acute exposures were based on the highest predicted one-hour-average concentrations. Predicted impacts at all receptors are below the significance criteria of 1.0; thus the project should have insignificant non-cancer health effects based on regulatory guidelines.

#### **5.16.2.4 Uncertainties in the Analysis**

Predictions of future health risks related to the proposed project are characterized by substantial uncertainties because of gaps in scientific knowledge in the practice of risk assessment, as well as the need to simplify some aspects of the process for a manageable computational effort. There are model and data uncertainties with respect to the assumed emissions, dispersion modeling and toxicological factors. There are also uncertainties with respect to the characteristics of the potentially exposed population. For example, parameters of possible exposure scenarios may include one or more of the following: that a person may be assumed to reside in one location for the average period of U.S. residency (about nine years); or for the 90th percentile of residency (about 30 years); or for an entire lifetime (about 70 years); and that exposure may be assumed at the highest modeled concentration, or some average, or a modestly high concentration representative of the exposed population.

Because risk assessments are often performed to set some regulatory limit on exposure in order to protect the public health, the assumptions of risk assessment have tended to more likely overestimate risk rather than underestimate it. The risk assessment methodology described above followed the CAPCOA *AB2588 Risk Assessment Guidelines* (CAPCOA, 1993), which are designed by regulators to more likely overestimate than underestimate health risks. The following discussion provides qualitative assessments of the uncertainties and variabilities in the major areas of an air toxics health risk assessment.

**5.16.2.4.1 Emissions.** The emission factor estimates for the gas turbine and obtained from the SCAQMD may be overly conservative due to the limited source test data used to derive these factors. However, for both the one-hour and annual averaging periods, it was assumed that the combustion turbine and the auxiliary boiler were operated at maximum load conditions. Also, the annual averaging period used maximum operation for 8,760 hours per year. Under actual operations, the hours of operation and typical heat input rates will be lower. The chemicals modeled were those with toxicity criteria in the OEHHA and CAPCOA

risk assessment guidelines, which are considered to be reasonably representative of commonly encountered air toxics.

**5.16.2.4.2 Air Dispersion Modeling.** In general, EPA-approved dispersion models such as ISCST3, tend to over-predict concentrations rather than under-predict them. For example, all chemical emissions are assumed not to be transformed in the atmosphere. For certain pollutants, conversion may occur sufficiently fast to reduce concentrations from the conservative model predictions. Moreover, these models use assumptions about plume dispersion that tend to over-predict concentrations.

**5.16.2.4.3 Exposure Assessment.** The most important uncertainties related to exposure concern the definitions of exposed populations and their exposure characteristics. The choice of a maximally exposed individual (MEI) is very conservative in the sense that no real person is likely to spend 24 hours a day, 365 days a year, over a 70-year period, at exactly the point of highest toxicity-weighted annual average air concentration. The greatest true exposure is likely to be at least 10 times lower than that calculated using the MEI assumption.

**5.16.2.4.4 Toxicity Assessment.** The final area of uncertainty is in the use of toxicity data in risk estimation. Estimates of toxicity for the health risk assessment were obtained from the OEHHA *Technical Support Document for Describing Available Cancer Potency Factors* (Cal-EPA, 1999a), OEHHA's *The Determination of Acute Reference Exposure Levels for Airborne Toxicants* (CalEPA, 1999b), OEHHA's *The Determination of Chronic Reference Exposure Levels for Airborne Toxicants* (Cal-EPA, 2000a; 2000b; and 2001), and the CAPCOA Air Toxics "Hot Spots" Revised 1992 Risk Assessment Guidelines (CAPCOA, 1993), which are among the most conservative compilations of toxicity information. Toxicity estimates are derived either from observations in humans or from projections derived from experiments with laboratory animals. Human data are obviously more relevant for health risk assessments, but are often uncertain because of: difficulty in estimating exposures associated with the health effect of interest; insufficient numbers of people studied; relatively high occupational exposures (the source of most human data), which must be extrapolated to low environmental exposures; or because the population being studied is more or less susceptible than the population as a whole. Cancer risk coefficients from human data are typically considered best estimates and are applied without safety factors. Cancer risk is typically considered proportional to pollutant concentration at any level of exposure (i.e., a linear, no-threshold model), which is conservative at low environmental doses. For non-cancer effects, the lowest exposure known to cause effects in humans is usually divided by uncertainty or safety factors to account for variations in susceptibility and other factors. When toxicity estimates are derived from animal data, they usually involve extra safety factors to account for possibly greater sensitivity in humans, and the less-than-human-lifetime observations in animals. Overall, the toxicity assumptions and criteria used in the proposed MPP's risk

assessment are biased toward overestimating risk. The amount of the bias is unknown, but could be substantial.

#### **5.16.2.5 Criteria Pollutants**

Four criteria pollutants were modeled and evaluated for their impacts on air quality and human health (see Section 5.2). Modeling of nitrogen dioxide (NO<sub>2</sub>), carbon monoxide (CO), sulfur dioxide (SO<sub>2</sub>), and particulate matter less than 10 micrometers in aerodynamic diameter (PM<sub>10</sub>) indicates that health impacts of criteria pollutants are not significant. Maximum predicted concentrations of the criteria pollutants were compared with National and California Ambient Air Quality Standards (NAAQS/CAAQS), which are health-based levels that serve as inhalation reference doses. With the exception of PM<sub>10</sub> and CO, which already exceeds the CAAQS, the NAAQS/CAAQS are not exceeded in the project area. Therefore, significant adverse health effects are not anticipated.

#### **5.16.2.6 Public Health Risks - Chemicals Stored and Used on Site**

The SCR air pollution control system would require the storage of aqueous ammonia in amounts exceeding the threshold planning quantity (TPQ) for the CalARP. This would be the only chemical that is considered to be an acutely hazardous material stored and used on site in amounts exceeding TPQs, and subject to RMP requirements under the CalARP regulations. Accidental releases of ammonia pose the potential to adversely affect public health. Refer to Section 5.15 (Hazardous Materials Handling) for more information and an assessment of potential offsite consequences. In summary, the offsite consequence analysis that was performed indicates that no significant offsite hazards would occur from an accidental release of aqueous ammonia (19% concentration).

The Applicant will coordinate with local emergency response units by: 1) providing them with copies of the plant site Emergency Response Plan; 2) conducting plant site tours to point out the location of hazardous materials and safety equipment; and 3) encouraging participation in annual emergency response drills.

#### **5.16.2.7 Summary of Public Health Risk Impacts**

Results from an air toxics risk assessment based on emissions modeling indicate that there would be no significant incremental public health risks from the construction or operation of the MPP. Results from criteria pollutant modeling for routine operations indicate that potential ambient concentrations of NO<sub>2</sub>, CO, SO<sub>2</sub>, and PM<sub>10</sub> meet federal requirements that have been established to protect public health, including the more sensitive members of the population.



### **5.16.3 Electromagnetic Field Exposure**

#### **5.16.3.1 Introduction**

Exposure to both electric and magnetic fields (EMFs) occurs where electric charges exist. Electric fields exist when these charges are not moving. Magnetic fields are created when the electric charges are moving. The magnitude of both electric and magnetic fields fall off rapidly as the distance from the source increases.

Transmission lines, distribution lines, house wiring, and appliances generate electric fields in their vicinity because of unbalanced electrical charge on unshielded energized conductors. Electric fields are expressed in volts per meter (V/m) or kilovolts (thousands of volts) per meter (kV/m).

Once electric currents are in motion, they create magnetic fields. The strength of the magnetic field is proportional to the magnitude of the current in the circuit. Magnetic fields can be characterized by the force they exert on a moving charge or on an electrical current. A magnetic field is a vector quantity that is characterized by both magnitude and direction. Electric currents are sources of magnetic fields. Magnetic fields are measured in milligauss (mG).

At the ground under a transmission line, the electric field is nearly constant in magnitude and direction over distances of a few meters. However, in close proximity to the transmission or distribution line conductors, the field decreases rapidly as distance from the conductor increases. Similarly, near small sources such as appliances, the field is not uniform and falls off even more rapidly with distance from the device. If an energized conductor is inside a grounded conducting enclosure, then the electric field outside the enclosure is zero and the sources is said to be shielded.

Concern about health effects from EMFs arose in 1979 when researchers calculated a weak statistical link between proximity to power lines and childhood leukemia. This study was based on wire-code classifications for residences and the incidence of leukemia. Since then, other researchers have investigated this potential association and other types of potential human health effects from EMFs.

In January 1991, the CPUC issued an Order Instituting Investigation (I.91-01-012, CPUC 1991) into the potential health effects from electric and magnetic fields emitted by electric power and cellular telephone facilities. In September 1991, the assigned CPUC Administrative Law judge issued a ruling that created the "California EMF Consensus

Group.” This group of representatives from utilities, industry, government, private and public research, and labor organizations submitted a document entitled “Issues and Recommendations for Interim Response and Policy Regarding Power Frequency EMF’s” on March 20, 1992 (California EMF Consensus Group, 1992). Regarding the relevant policy consensus recommendation titled “Facility Siting,” the group stated that the CPUC should recommend that utilities take public concern about electromagnetic fields into account when siting new electric facilities. Although this group could not conclude that there is a relationship between EMF and human health effects, they also could not conclude that this relationship does not exist to any extent; therefore, they recommended that the CPUC authorize further research.

In 1991, Congress asked the National Academy of Sciences (NAS) to review the research literature on the effects of EMF exposure and determine whether sufficient scientific basis existed to assess health risks from such exposure. In response, the National Research Council (NRC) convened the Committee on the Possible Effects of Electromagnetic Fields on Biologic Systems. After examining more than 500 studies spanning 17 years of research, the committee concluded in an October 1996 report that there is no conclusive evidence that EMFs play a role in the development of cancer, reproductive and developmental abnormalities, or learning and behavioral problems (NRC, 1996).

On June 27, 1998, a 28-member advisory panel sponsored by the National Institute of Environmental Health Science (NIEHS), part of the National Institute of Health, voted 19 to 9 to label EMFs a “possible human carcinogen,” which kept open funding for continuing government studies. On May 4, 1999, NIEHS issued a report entitled *Health Effects from Exposure to Power-Line Frequency Electric and Magnetic Fields* (NIEHS, 1999). This report found that the evidence is “weak” that electric and magnetic fields cause cancer. The report concludes: “The NIEHS believes that the probability that EMF exposure is truly a health hazard is currently small. The weak epidemiological associations and lack of any laboratory support for these associations provide only marginal scientific support that exposure to this agent is causing any degree of harm.” While the report says EMF exposure “cannot be recognized as entirely safe,” the report goes on to say “... the conclusion of the report is insufficient to warrant aggressive regulatory action.” Because virtually everyone in the United States is exposed to EMF, the report recommends that “... passive regulatory action is warranted such as continued emphasis on educating both the public and the regulated community on means aimed at reducing exposures,” but that cancer and non-cancer health outcomes do not provide “... sufficient evidence of a risk to warrant current concern.”

### **5.16.3.2 Project Impacts**

Section 3.6 described the proposed transmission line from the MPP to the Olive Switchyard as an underground 69-kV transmission line with shielded cable. The use of shielded cable located 48 inches below the ground will not produce any electric fields at the surface. Magnetic fields can penetrate the ground and occur at the surface along the proposed underground transmission lines. However, these magnetic field strengths are expected to be minimal and the areas along the cable route will not be accessible by the public. Therefore, public exposures to magnetic fields from the proposed 69-kV tie are projected to be insignificant.

Appendix Q presents the results of the Interconnection Study with potential connections to existing 12.5-kV, 13.8-kV, 34.5-kV and 69-kV lines and transformers on the COB and Glendale systems, and existing 230-kV and/or 500-kV lines on the LADWP and Edison systems. The Interconnection Study concluded no negative impacts on any of these existing systems. That is, these existing systems have capacity to accommodate the proposed 250 MW MPP Project. The study did note the potential for overload on the proposed 69-kV interconnection ties, which could be addressed by installing larger underground lines or reducing the capacity of the Project. Since larger lines would still be underground and shielded, electric field exposures would still be zero. Magnetic field exposures would still be minimal.

Potential EMF increases along the existing COB, Glendale, LADWP and/or Edison transmission routes would represent a small increase over current EMF levels. California does not currently have regulatory levels for transmission line electric and magnetic field strengths. States with regulations range from 1.0 kV/m to 2.0 kV/m for electric fields at the edge of the right of way to 11 kV/m within the right-of-way, and 150 mG to 250 mG for magnetic fields at the edge of the right of way, depending on voltage. Current EMF levels along the existing transmission systems are anticipated to be well within these limits, and the proposed project would add fractionally to existing levels. Given this and the lack of sufficient evidence of health hazards to exposed humans, there is no anticipated impact on public health. Although the public health significance of project-related exposures cannot be characterized with certainty, the current evidence in the scientific literature suggests that such risks, if any, would be small. Any long-term exposures are estimated to be within normal background levels.

### **5.16.4 Mitigation Measures**

The proposed project has been designed to minimize potential public health risks, including use of natural gas as fuel, and incorporation of appropriate emission control measures. Based

on the results of the air toxics risk assessment, no additional mitigation measures are required to reduce risks, since all risk estimates are well within acceptable levels. Because electric and magnetic field strengths are expected to be within normal background levels, no additional mitigation measures are required.

#### **5.16.5 Significant Unavoidable Adverse Impact**

No significant unavoidable adverse impacts on public health are anticipated from the proposed project.

#### **5.16.6 LORS Compliance**

LORS that are applicable or potentially applicable to the MPP in the context of public health are outlined in Section 7.4.2. The MPP will operate in accordance with all LORS applicable to public health.

#### **5.16.7 References**

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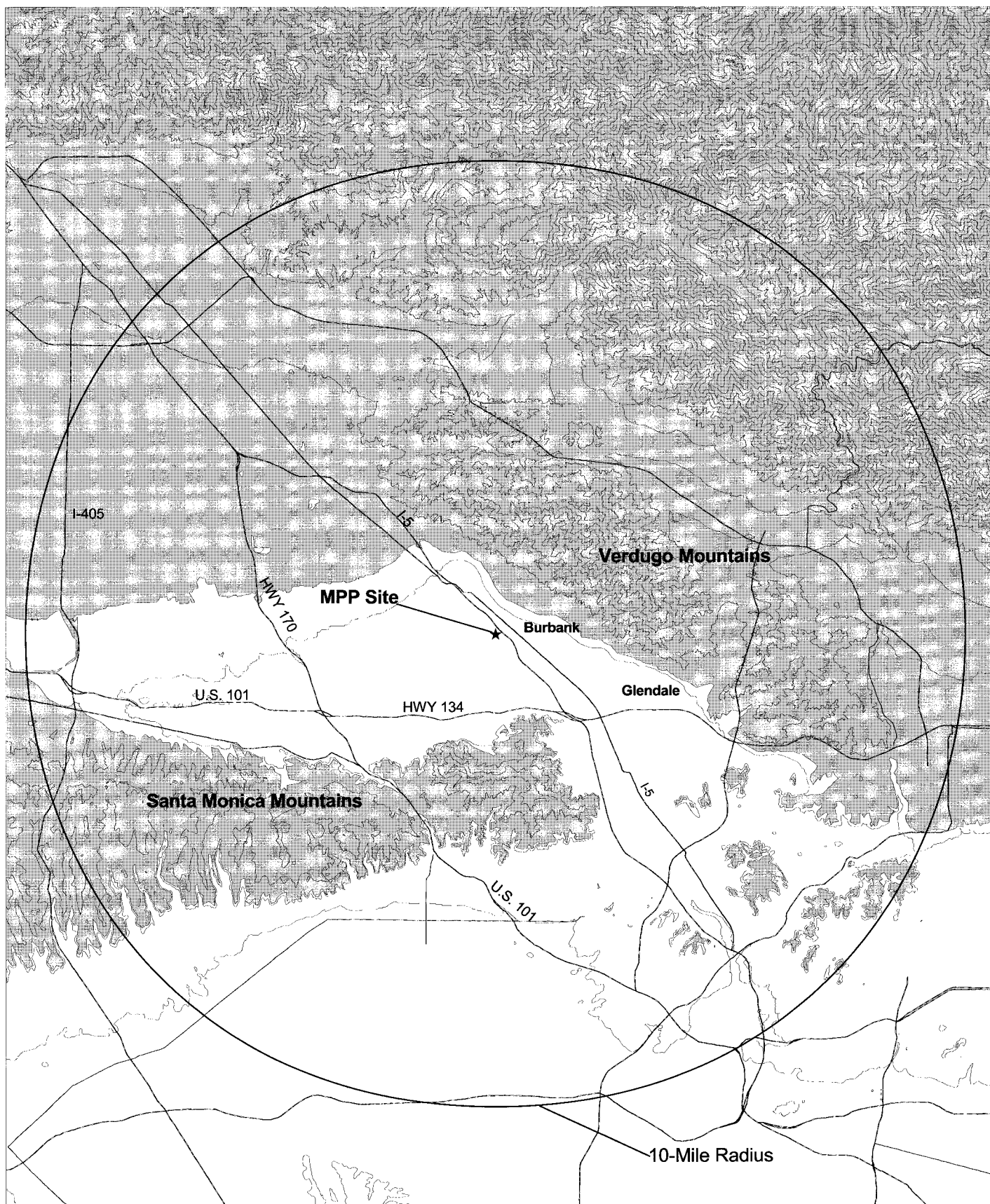
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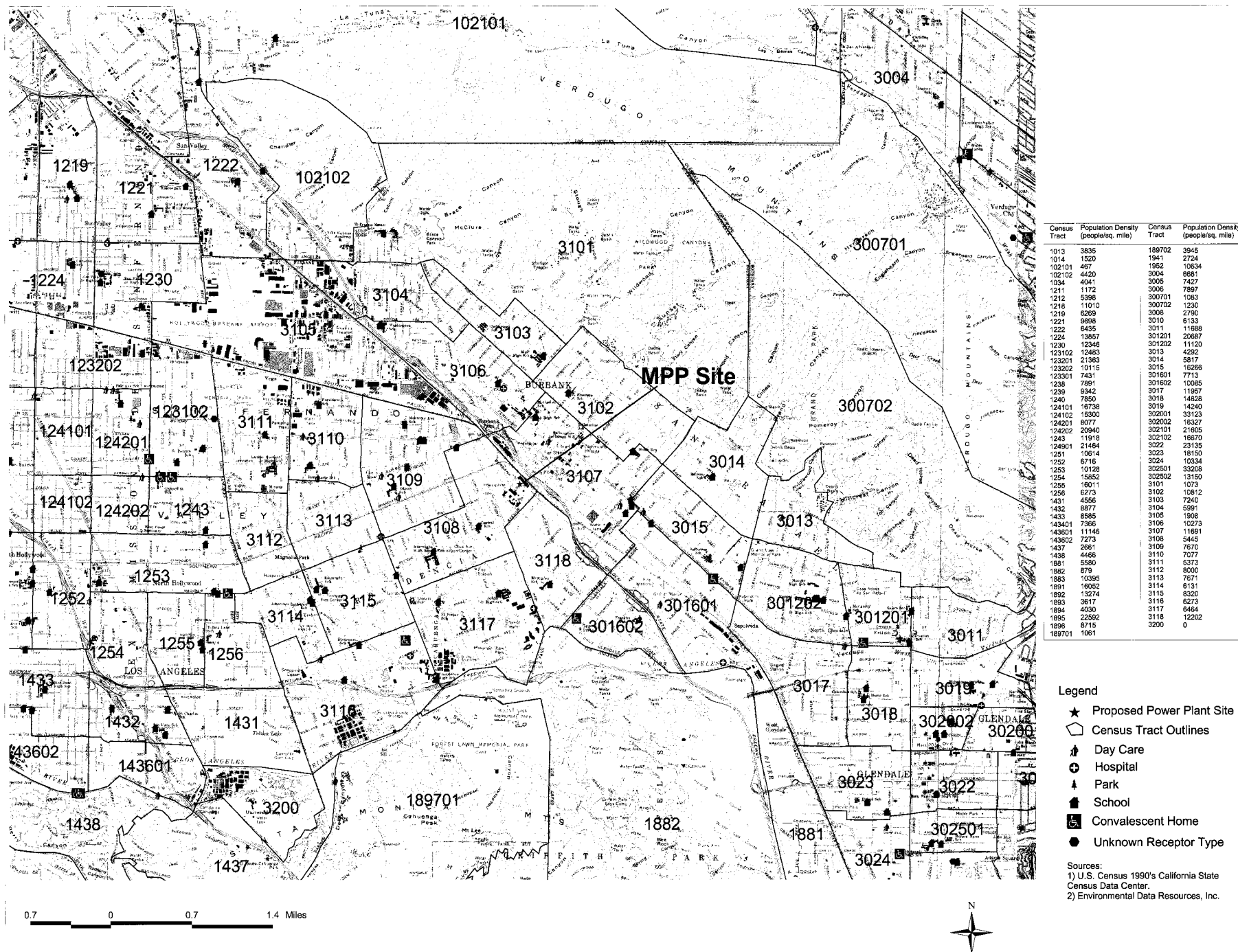
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Magnolia Power Project Figure 5.16-2. POPULATION CENSUS TRACTS, POPULATION DENSITY, AND SENSITIVE RECEPTORS

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